Massive Tracheal Necrosis Complicating Endotracheal Intubation

Nancy C. Abbey, M.D.; Dale E. Green, M.D.; and Michael J. Cicale, M.D., F.C.C.P.

There are significant complications associated with endotracheal intubation. Massive tracheal necrosis secondary to tracheoesophageal space abscess developed in a 71-year-old man during mechanical ventilation. Elevated endotracheal tube cuff pressures, sepsis, hypotension, and other risk factors predispose to this disastrous consequence. (Chest 1989; 95:459-60)

Complications of endotracheal intubation are well described in the medical literature and include sore throat, laryngitis, glottic edema, mucosal ulceration, laryngeal stenosis, tracheal stenosis, tracheal dilatation, unrecognized esophageal intubation, and tracheal-innominate artery fistula.1,2 We present a case of tracheoesophageal space abscess complicated by massive tracheoesophageal fistula.

CASE REPORT

A 71-year-old man was admitted to an outside hospital for fever and neutropenia. On examination, the blood pressure was 150/80 mm Hg and the oral temperature was 38.6°C. There was an uncomplicated rectal fissure. The hematocrit value was 41 percent and the leukocyte count was 600/cu mm with no granulocytes. Blood cultures grew Pseudomonas aeruginosa and an antibiotic regimen was begun. Bone marrow aspiration revealed absence of granulocyte precursors. The agranulocytosis was attributed to a drug reaction from chlorhalidone. Over the next week, the patient had increasing dyspnea and developed respiratory failure associated with bilateral pulmonary infiltrates. There were intermittent episodes of hypotension secondary to supraventricular tachyarrhythmias. He was intubated and mechanically ventilated. The leukocyte count returned to normal (9,600/cu mm) after one week; however, the rectal fissure developed into a perirectal abscess. This was treated with saline irrigations. Because of rising airway pressures, the endotracheal tube cuff pressure was increased to prevent air leakage. Ten days after intubation, tracheostomy was performed. Upon skin incision, the endotracheal tube was found to be in a pocket of pus. The entire circumference of the trachea was necrotic with the esophagus forming the posterior wall. The distal tracheal stump extended approximately one-half inch above the carina and was surrounded by pus. In the chest, the innominate artery formed the anterior wall of the tracheal passage. A skin flap was placed between the tracheal space and the innominate artery. Drainage tubes were placed in the mediastinum. An endotracheal tube was placed into the distal tracheal stump and secured with sutures. Postoperatively, the patient had concomitant insufflation of the stomach with each ventilator cycle. A gastrostomy tube was placed for decompression. The patient was transferred to Shands Teaching Hospital. On admission, the patient was hemodynamically stable and afebrile. He was awake and followed simple commands. Auscultation of the lungs revealed bilateral coarse rhonchi. There was a large perirectal abscess. Laboratory studies revealed a white blood cell count of 12,200/cu mm, with 91 percent granulocytes. The arterial blood gas values obtained on a fractional inspired concentration of oxygen (FIO2) of 45 percent were a pH 7.35, a partial pressure of carbon dioxide (PaCO2) of 43 mm Hg, and a partial pressure of oxygen (PaO2) of 80 mm Hg. The admission chest x-ray film revealed diffuse bilateral infiltrates. Computed tomography of the chest revealed destruction of the trachea from a level 2 cm distal to the true vocal cords to approximately 3 cm above the carina. The tip of the tracheostomy tube appeared to be eroded through the posterior wall of the trachea into the esophagus (Fig 1). Blood cultures and cultures of pus from the peritracheal abscess subsequently grew Pseudomonas aeruginosa. The patient was treated with broad spectrum antibiotics to which the Pseudomonas was sensitive. The perirectal abscess was treated with saline irrigation.

Because of air escaping around the endotracheal tube, adequate ventilation was an increasing problem. The single endotracheal tube was removed and replaced with separate tubes selectively intubating each mainstem bronchus and connected to separate ventilators which fired synchronously. Despite continued treatment with antibiotics and respiratory support, the patient died three weeks after admission.

A post-mortem examination revealed a tracheoesophageal fistula 8 cm in length. It extended from the larynx to 3 cm proximal to the carina (Fig 2). The lungs revealed diffuse alveolar damage and bilateral necrotizing pneumonia.

DISCUSSION

The incidence of major tracheal complications secondary to endotracheal intubation ranges from 0.3 to 19 percent.3,4 These major complications include tracheal stenosis, tra-

*From the University of Florida College of Medicine, Department of Medicine, Division of Pulmonary Medicine, and Gainesville Veterans Administration Medical Center, Gainesville, FL.
+Christmas Seal Fellow, American Lung Association of Florida.

Figure 1. CAT scan of upper thorax: 1, sternum; 2, arch of aorta; 3, abscess cavity; 4, endotracheal tube; and 5, vertebral body.
choesophageal fistulas, tracheoinnominate artery fistulas, and cuff trachietasis. We could find no case of this severe complication in the current literature.

A number of factors have been postulated to contribute to tracheal damage associated with tracheal intubation. Elevated endotracheal tube cuff pressure is a common predisposing factor to tracheal damage. One study documented a 19 percent incidence of cuff pressures greater than 25 mm Hg and an 11 percent incidence of cuff pressures greater than 60 mm Hg. Increased cuff pressures are often used in patients with decreased lung compliance to maintain air seal during mechanical ventilation. Knowlson and Bassett postulated that a cuff pressure above 32 mm Hg would exceed the perfusion pressure of the tracheal mucosa and would result in pressure necrosis. Autopsy studies have shown a consistent pattern of progressive damage starting with superficial trachietitis followed by ulceration, and finally fragmentation and dissolution of cartilaginous rings. Microscopically, acute inflammation and hemorrhage lead to ulceration and ultimately, perichondritis. Laryngotracheal injury in autopsy specimens postintubation can be as high as 95 percent.

Hypotension predisposes to tracheal necrosis presumably by decreasing capillary perfusion around the endotracheal balloon cuff. Systemic and respiratory infections have also been associated with an increased risk of tracheal damage. The damaged tracheal mucosa may be more susceptible to bacterial infection from contaminated secretions or hematogenous seeding. Other risk factors implicated are nasogastric tubes, patient agitation and the type of endotracheal tube used.

This case illustrates a dramatic complication of endotracheal intubation. This patient had many risk factors often associated with tracheal necrosis including pneumonia, sepsis, decreased lung compliance secondary to adult respiratory distress syndrome, and hypotension. A nasogastric tube was present during much of the hospitalization. The patient was frequently agitated and at times required sedation and paralysis. All of these factors probably initiated trachietis, and later, tracheal necrosis and formation of a tracheoesophageal fistula.

The signs and symptoms that tracheal damage is occurring are subtle. The inability to maintain an adequate seal with the endotracheal tube cuff is the most obvious sign. Perhaps most importantly, a high index of suspicion must be maintained in all intubated patients. Daily cuff pressure monitoring, correction of hemodynamic abnormalities, sedation, adequate antibiotics, and avoidance of tissue anoxia will help prevent this type of injury.

Treatment of tracheal defects has been accomplished in some circumstances. However, the longest section of trachea successfully replaced was 5 to 6 cm. The degree of necrosis and infection in addition to the patient's inability to be weaned from ventilatory support precluded any attempts at repair.

In conclusion, we found this case to be a dramatic example of the possible complications of endotracheal intubation. Meticulous attention needs to be given to the care of endotracheal tubes in critically ill and debilitated patients to avoid these tragedies.

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